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BRADYKINETIC ANALYSIS OF SOMATIC MOTOR DISTURBANCES IN NERVOUS DISEASES

ANALYSIS OF MOTOR DISORDERS BY THE AID OF ULTRA-RAPID MOVING PICTURES

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BRADYKINETIC ANALYSIS OF SOMATIC MOTOR DISTURBANCES IN NERVOUS DISEASES

ANALYSIS OF MOTOR DISORDERS BY THE AID OF ULTRA-RAPID MOVING PICTURES

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The subject of disturbed motor function has been carefully studied for many years with some interesting results especially in the clinical field. Motor defects, however, have proved elusive to exact interpretation and in many respects knowledge in this particular field is still in need of assiduous cultivation. It seems probable that the principal difficulty lies in the present imperfect understanding of vertebrate motion as a whole, and in this light the interpretation of disorders in the complex motor mechanisms of man appears all the more perplexing. It has been apparent to the authors and others interested in this important topic that only by a combination of many different methods may the problems involved in the motor disturbances be solved. The object of this paper, therefore, is to present certain newer methods of study which may be applied to these disorders, and at the same time to demonstrate the results derived by an application of the methods to certain well-defined groups of motor disorganization.

Weisenburg, a few years ago, demonstrated the value of cinematographic reproduction in the study of nervous diseases. In the present investigation more recent developments in photography with the high speed camera are introduced in the analysis of deformities in motion.

In an attempt to analyze and record the disturbances of motility, one is impressed at the outset by the inadequacy of the nomenclature employed in describing various types of abnormal motion. Most of the terms in common use not only fail to be descriptive, but lexicologically many of them are nondescripts. For the present and until adequate replacements in terminology may be made, it seems desirable to adhere to the accepted nomenclature.

METHOD AND MATERIAL

Ultra-rapid moving pictures of patients by means of the "Novograph" camera were taken at the rate either of one hundred and sixty or three hundred exposures per second. The ordinary cinematographic rate is sixteen exposures per second, so that the ultra-rapid rate is from ten to eighteen times as fast. The normal rate of projection on the screen offers opportunity for the study of every component in each movement. The motion, thus slowed down, serves as the basis for what we have called *bradykinetic analysis.*

In studying each of these types photographs were made simultaneously at the usual and also at the ultra-rapid rate of exposure. The screen reproduction of these pictures first shows the subjects photographed at the regular cinematographic rate, followed by the pictures taken at the ultra-

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rapid rate, both sets of photographs being reproduced at the usual cinematographic rate of sixteen pictures per second.

The material upon which these studies were made consisted of patients selected from the neurological wards of the Montefiore Hospital for Chronic Diseases. In this selection special attention was given to securing patients showing marked motor disorders the general characters of which have been the subject of much debate and have also occasioned not a little discussion in classification and nomenclature. The motor disorders submitted to bradykinetic analysis include choreiform movements, torsion spasms, extreme intention tremors and cerebellar ataxia. These motor defects occurred in cases of *chronic degenerative chorea, astasia abasia, dystonia musculorum deformans, multiple sclerosis* and *congenital cerebellar aplasia.*

In all the cases studied the motor defect was characterized by some pronounced abnormal and adventitious element of motion which produced a definite deformity of movement. Such adventitious motor elements we have endeavored to describe as objectively as possible in the following enumeration of them:

1. Regular and rhythmical oscillations of parts of the body during muscular inactivity or in volitional action.
2. Irregular arrhythmical oscillations of parts of the body during volitional motion.
3. Sudden isolated twitches of the muscles producing involuntary movements or deflecting the course of volitional motion.
4. Sudden successive twitches of muscles producing involuntary movements or deflecting the course of volitional motion.
5. Sustained mobile spasms of muscles during volitional motion producing rhythmical distortions of the limb, trunk or limited parts of the body during activity of the particular part.
6. Sustained tonic spasms of limited groups of muscles during volitional motion which retard or deform the resulting movements.
7. Sustained tonic spasm in a large number of muscle groups during activity or in rest, producing distortion of the body together with marked deformity of motion during action.

I. ANALYSIS OF A CASE OF CHRONIC DEGENERATIVE CHOREA

This patient was a married woman, thirty-six years of age, born in Russia. She was the mother of five healthy children. Her present illness began when she was thirty-one years old during the fifth month of a pregnancy. Without apparent cause there developed irregular, purposeless, involuntary movements in all segments of the body. A gradual change in conduct was also observed which apparently affected the faculties of attention and concentration. She showed lack of interest in her family and in affairs generally, with a progressive mental deterioration.

The cinematographic study of the patient at the normal rate of projection showed a variety of meaningless and purposeless deformities of motion such as facial grimacing, continuous movement of the eyeballs together with sudden twitchings in the trunk, limbs and head. These movements were somewhat too rapid for the typical Huntington's chorea. There was incessant jaw motion as in grinding the teeth, and the tongue and lips smacked at irregular intervals. The abdominal muscles were in almost constant play.

By ultra-rapid photography, the projection revealed striking differences for interpretation. The component elements of the deformities of motion were clearly discernible, and what appeared to be twitching, purposeless movements were seen to have definite associative significance. They comprised a series of symbolic attitudes indicative of emotion. The photographic analysis reduced the speed of movement and resolved the motions of the trunk and extremities into segmental components. These movements now appeared as those of approach and retreat, expressing the appeal of entreaty or negative self-feeling of shame. They clearly represented an emotional flux recognized as a functional element in some patients with this type of chorea.

The posing and attitudinizing, so impressive in other well-recognized organic diseases, suggest a close anatomical relation of these symbolic movements to the basal ganglia of the forebrain. The thalamus and the corpus striatum which are functionally associated with sensorimotor complexes serve as the centers especially controlling automatic associated acts and attitudes expressing the fundamental emotions. In this light it seems fair to presume that such attitudes as express emotional states in true hysteria may have their subconscious controlling level in these basal ganglia.

Another interesting feature in the bradykinetic analysis was the extreme hyperextension of the left leg at the knee as the patient walked. In this patient the attitude of the limb appeared to be the result of kinetic overflow within the extensors, the check of the antagonistic flexors being defective. The extreme slowness of the antagonistic check response together with a singular wave reaction seen in the extensors suggested a possible failure of elements constituting the so-called static mechanism. A similar failure in stabilizing the kinetic flow is seen in the study of the dystonias, and the imbalance in the synergic units points to a defect in the synergizing influence of the cerebellar reflex circuit.

In summary, the analysis of this case of degenerative chorea discloses a combined defect, in part affecting the kinetic mechanism controlling automatic associated movements and attitudes expressive of the emotions, in part due to impairment in the static mechanism maintaining posture. (Fig 1.)

II. ANALYSIS OF A CASE OF CHRONIC DEGENERATIVE CHOREA WITH SOME ELEMENTS SUGGESTIVE OF DYSTONIA

The patient was thirty-six years of age. About two years previous he became "extremely nervous" and developed slight twitching in the lower extremities, soon accompanied by a staggering and uncertain gait. Four months later the upper limbs participated in motor restlessness, consisting of involuntary "flinging" about of the arms followed by shrugging movements of the shoulders. The same disturbance involved muscles of the head and neck. A corkscrew-like motion, chiefly of the trunk, completed the motor disorder.

When admitted to the Montefiore Hospital he had the attitude, movements and gait of Huntington's chorea.

By bradykinetic analysis the attitudinizing and symbolization, so evident in the first case, were apparently distinctly those of hurling or lifting and were too definite in pose and in poise to be a mere chance overflow of kinetic energy into purely accidental designs. Unquestionably there was an element of purpose in these attitudes. The postures were those of aggression and attack, thus expressing what may be expected of the more positive character of the male emotive mechanism. An extensor elevation of the foot (not seen in the accompanying photographic illustrations) as the patient stepped forward, and also a corkscrew-like movement of the trunk upon the pelvis, appeared to be distinctly dystonic elements and suggested a relationship between the dystonias and this type of chorea.

In all the cases presenting clinical features that seem to involve the posturing and attitudinizing mechanism, the motor elements which determine posture seem to be disorganized. In cases of this type the agonist and antagonist are apparently not harmoniously cooperative. This becomes manifest as the several muscles are studied in their slow interplay. Comparison by means of bradykinetic analysis readily demonstrates a marked difference between disturbances due strictly to cerebellar disintegration of the synergic unit, and those forms of striatal disintegration whose pathological anatomy probably lies in the corpus striatum and its immediate basal connections, especially with the red nucleus.

As many observers have pointed out, there is doubtless an intimate anatomical pathological relationship between the various deformities of motion of the striatal type, and in all of our cases a study of the muscle-group activities brings clinical confirmation of this fact.

The prolonged pronation spasms in this patient and the marked overaction of the extensor muscles holding the raised foot upon the toes, clearly belonged to the group of sustained mobile spasms producing rhythmical distortions of the limbs and body during motion. This phenomenon is particularly prominent in the dystonias.

The tendency of trunk distortion was disclosed as a sudden unilateral pull of flexor trunk groups. The torsion in this case involved rather the upper trunk segment in contrast to that seen in dystonia. There was a suggestion, too, of tic element in the choreic movements which involved especially the axio-appendicular groups of the upper extremities. Analysis certainly demonstrates a variety of elements common to many entities. (Figs. 2 and 3.)

In summary, the analysis of this case of degenerative chorea discloses the fact that sudden isolated twitches of the muscles producing involuntary movements or deflecting the course of volitional motion are to be associated with disturbance in the striatal and cerebellar mechanisms, and that the attitudinizing, although different from that observed in the woman suffering from the same disease, is basically similar in being expressive of primitive emotions. In the case of the male these emotions have a positive self-feeling tone representative of aggression and attack in contrast to the female where these emotions have their expression as negative self-feeling tone in reactions of appeal and retreat. The sustained tonic spasms producing distortions are manifestations of hyperkinetic innervation of muscles recognized as the essential part of the syndrome in those motor disorders known as the dystonias.

III. ANALYSIS OF A CASE OF MULTIPLE SCLEROSIS WITH ASYNERGIC MOVEMENTS

The patient, a watchmaker, forty-eight years of age, first noticed, when he was thirty-two years old, a slight and occasional defect in vision, with numbness in his fingers which occurred from time to time. A year later he began to be troubled with a tremor in his right hand especially when executing the more delicate manipulations of his trade. This tremor gradually spread until it involved his entire body. He soon developed difficulty in walking, and in the course of three years his locomotion was so much compromised by a spastic paraplegia that he was forced to give up his occupation. It was about this time that the nature of his malady was recognized.

The study of this case at normal photographic speed discloses many confusing movements as the subject attempts the "finger-to-finger" approximation test. He illustrates particularly well a phenomenon which, in part, characterizes the motor disturbances of disseminated sclerosis, the so-called *intention tremor.* These abnormal movements during voluntary effort were, from an early period in the history of his disease, very extensive, and comprised, indeed, the only symptom present for some time. Original observers of the case placed it among functional disorders.

The analytic projection shows a disintegration of the synergic units throughout the body. Agonist and antagonist are not coordinated. The extensor check fails to hold the flexors and the reverse is also true.

This disorganization is especially obvious in the finger-to-finger test as the point of destination and finer approximation is approached. In slow motion the disproportion in flexion, extension and supination is clearly seen. The arms shoot far beyond the point desired (the analytic photography greatly reducing the speed), they are overextended and again overflexed. The obtrusive feature is a motor disorganization showing dissociation of muscular harmony and asynergia between dominant and check groups of muscles. As has been shown by Weisenburg and Tilney, this type of motor phenomenon is indicative of cerebellar disturbance. The groups of muscles of the trunk and extremities are also involved. The case is one of integrative asynergia. It would seem then that the "tremor" of multiple sclerosis is of cerebellar origin, that a case such as the one studied must involve extensive cerebellar areas. (Fig. 4.) An even more definite conception of cerebellar asynergia is acquired by a study of a case of cerebellar agenesis.

In summary, the analysis of this case of multiple sclerosis reveals the fact that the so-called intention tremor is a static defect due to the dissociation of the synergic units of the body consequent upon disproportionate innervation of the dominant and check elements in the muscle groups.

IV. ANALYSIS OF A CASE OF ASTASIA ABASIA WITH EXCESSIVE ISOLATED AND SUCCESSIVE SPASMS

The patient was a young girl, seventeen years old, apparently with nothing in her family history to cause the onset of a profound psychoneurosis. The abnormal movements consisting of isolated and successive muscular spasms had their exciting cause some four years previous in the fear of a thunder storm. Associated with these movements was a psychic attitude of dread with sudden increase of the motor agitation and facial expression of anxiety upon any sudden noise. Up to that time there had been no disturbance of consciousness nor had there been objective or subjective sensory symptoms of hysterical nature.

The motor manifestations were those of astasia abasia, the patient being unable to stand or walk without support.

At normal speed photography, as the patient stood and walked with support, the gait was not suggestive of any organic nervous disease. The segments of the body, as the muscles moved the joints, acted in harmony and unison; there was no disintegration of the synergic units. The lower extremities in walking performed a series of short steppage movements, and in standing these continued in rapid succession. Analyzed under increased photographic speed, rhythmic, well poised, fully coordinated, normally synergic phases of voluntary movement followed in regular order. Their contrast with the preceding studies of organic deformities of motion leaves no doubt of the different character of each. These movements clearly bespeak an intense self-consciousness with acute apprehension of impending danger. They are more complex and studied than the more direct instinctive reactions to fear. Their elaborate character is in marked contrast to the simpler attitudinizing in chorea.

Another muscular phenomenon was observed in this case, namely, a vermicular muscle wave. This was not the result of any artificial or mechanical effect and must have depended upon some inherent quality of the muscle groups. The muscle bellies themselves were apparently the seat of gross morphological changes. Whether this phenomenon is pathognomonic of functional motor disturbances can be determined only upon bradykinetic studies in many other cases. Mr. C. P. Watson, President of the Novograph Company, whose experience with screen photography is vast, and who had witnessed film productions of many athletes and others under varying conditions of muscular activity, had not previously observed this vermicular wave. In the normal projection of this girl there was no suggestion of this wave, but in slow motion analysis the wave was immediately recognized passing slowly from below upward through all the muscles of the right lower extremity from ankle to hip. This appearance resembles the successive surface movements when a strong wind sweeps a field of wheat. It would be of interest to study by bradykinetic analysis a number of functional disturbances in which the somatic musculature is involved. Possibly this wave as demonstrated in this patient is a condition found only in what we are wont to call non-organic or functional disorders.

Note: Some few weeks after the photographic presentation and following a fright, the patient suddenly passed into a kind of hypnotic sleep from which she could not be aroused even by the strongest stimuli, including faradic current of great intensity. In the beginning of this state the girl screamed continuously, had tonic and choreic convulsive seizures and assumed bizarre, emotional attitudes. She would constantly repeat, "I am afraid," but could not be aroused to any form of conscious response. There was corneal as well as general anesthesia. The condition continued for some three or four days. She was regularly tube-fed and catheterized. The irregular convulsive seizures were really a marked intensification of previous movements with an intense psychic phase added; they were so violent as to yield only to chloroform narcosis pushed beyond the primary stage. The patient at no time showed signs of exhaustion. There were several periods of these seizures, each lasting a few days. It seems to be of some significance that the patient was restored to consciousness by the playing of phonograph records. A more careful study of the intricate psychic associations was not made. The patient has since markedly improved psychically, but it is of interest to note that the toes in which intense flexor movements were observed on the screen have since assumed a definite and fixed attitude in the flexor position, though in sleep they at times become relaxed. (Fig. 5.)

In summary, the analysis of this case of astasia abasia reveals the fact that the irregular, sudden twitchings of the muscles which disturbed locomotion were in reality highly complex and expressive of acutely conscious and psychically much magnified fear. These movements are in marked contrast to the simpler attitudinizing of chorea. The wavelike contraction in the muscles of the leg requires further study before a complete analysis of it may be offered.

V. ANALYSIS OF THREE FAMILIAL CASES OF DYSTONIA MUSCULORUM DEFORMANS (A BROTHER AND TWO SISTERS)

We are indebted to Dr. I. Abrahamson for the opportunity of studying this series of cases.

In this family group of dystonia musculorum deformans, each member presented a different degree of severity and progress of the disease. The family stock was recorded as having been substantially without taint except in one important particular: On the paternal side there was a more or less direct history of dystonia, in that the patients' father's sister and her daughter were afflicted with a disease similar to that of the three patients here described.

1. *The Brother. Incipient Stage. Movements Largely Confined to One Extremity.* The boy was the least afflicted of the group. As in the case of the sisters, his early life up to the time of onset of the present illness was quite normal. The motor disturbance began in his thirteenth year, and as in the case of the younger sister, manifested itself especially in an attempt at execution of finer movements with the right hand, such as writing. The patient was intelligent and had made from the first every effort to overcome his difficulty. He fully realized the progress and serious deformity which the disease had shown in his sisters and was fearful, even before the onset of his illness, that he too might become a sufferer. Indeed, the boy made unusual efforts by exercise and intense mental endeavor to maintain control over the afflicted upper extremities; this he continued with great determination as he felt the right leg, more particularly the right foot, assume spasmodic, uncontrollable, involuntary attitudes in attempting movement.

The cinematographic representation of his gait showed a turning-out of the foot and the sudden flexion at the knee and hip with inward rotation at the hip. In normal projection as he ran, a limp alone was seen. The slow motion for bradykinetic analysis showed a partial failure of coordinate time reaction in groups of agonists and antagonists. A study of our series of dystonias shows this failure as a feature in them all. For example, the forceful hyperextension of the foot, common to all of the cases of dystonia, appears as a failure in the time limitation of the muscular contraction, or, in other words, an excessive overflow of innervation. The peculiar character of this kinetic overflow in the agonist groups differentiates the organic from the functional disease with which this latter spasmodic condition has often been confused. From the standpoint of muscular dynamics, this excessive muscular contraction is wasteful and unnecessary for the purposes served by the muscle action. A small percentage of energy is expended in the purpose and a large amount in producing useless tonic spasms of the muscles involved.

The slight rotation of the thigh seen on normal projection, together with a slight raising of the foot in equinus position and flexion at the knee and hip, was in reality a widespread disorganization of the movements of the gait. Analysis showed at once that this form of deformity was due to excessive overflow of kinetic innervation.

Any movement designed to serve some definite purpose, as for example, moving the leg forward in walking, may be considered as a transition from an *initial posture to terminal posture*. The leg may be arrested at any intermediate point and be made to assume an *intermediate posture*. The motion, therefore, from the initial to the terminal posture may be regarded as a series of potential intermediate postures between the two limits mentioned (initial and terminal posture). The innervation producing motion through a series of postures from the initial to the terminal is *kinetic,* while the innervation controlling the entire series of postures, both actual and potential, is *static*. In this light two streams of innervation flow concurrently and in harmony through the production of any purposive motion, the kinetic stream producing movement, and the static stream maintaining actual and potential posture. One or both of these currents may be subject to excessive innervation and produce excessive muscular activity in motion or in posture. Bradykinetic analysis indicates that both types of innervation are excessive in the dystonias with resultant extreme degree of movement and extreme postures amounting in many instances to actual distortions. In effect, therefore, dystonia is due to hyperkinetic and hyperstatic innervation. This is seen in this patient whose trunk tended to a rotary action about the pelvis while he was suddenly raised upon the toes of the right foot and thrown, as it were, as from a springboard. The extreme pronation of the upper right extremity gave the characteristic pose which in the more advanced stages assumes fixation. The torsion of the pelvis, commonly discribed in this disease, was only suggested in this patient, though a study in slow motion of the long muscles of the back and those about the pelvis showed the tendency to rotary motion.

In the study of the individual muscle groups by bradykinetic analysis, those of the right thigh stood out with abnormal intensity during periods which demanded their function only to a slight degree, thus indicating the hyperkinetic innervation. This appeared as a prolongation of muscular action. To a less degree this was conspicuous in the left thigh. It has been suggested that this latter may be an incipient phase of the characteristic deformities in the dystonias. The patient made every effort to overcome the deformity of movement, and in the early stages of this disease there seems to be no question that volition exercises some beneficial influence. Indeed, in a case seen in its incipient stages by one of us (Goodhart) and shown at the New York Neurological Society, exercises, relaxation and re-education added their measure of influence in causing the deformities to disappear entirely. The patient occasionally, and only when extremely tired, shows a momentary dystonic spasm. In the advanced stages, however, voluntary effort at control intensifies the deformities of motion. (Fig. 6.)

2. *One Sister. Dystonia Movements More Extensive and Intense.* The patient, an intelligent girl of eighteen, was the second in this family of three to become a victim of dystonia. Her condition represented a degree of involvement between that of the slightly affected brother and the more advanced case of another sister. There was no retardation in early development, the three children of this family having begun to walk and talk before the age of two.

When twelve years old the patient noticed a sudden jerking or forced movement in the form of pronation, beginning in the right hand. This motor difficulty showed definite advance in the performance of skilled movements generally, particularly in writing, and there were peculiar stiffening and worm-like movements in the fingers which seemed to extend suddenly upon attempts to grasp objects. The left hand soon became similarly affected. Within a few months the right leg showed some disturbance of motility. Gradually a feeling of tension developed in the leg, the extremity became flexed and the hip more or less fixed in this position (Fig. 7). As in all of these cases of dystonia musculorum deformans, this patient became easily exhausted upon attempts at voluntary movements. This is doubtless due in large measure to the efforts at suppression and the extra endeavor to use muscle groups, some of which are already involved. The relation of the trunk and pelvis was such as to cause lordosis of the lower dorsal and lumbar spines, and the entire body assumed an attitude of emprosthotonos. The characteristic hyperextension of the foot was present and a limp much more pronounced than in the brother. There was a marked likeness in the poise and position of the three members of this group. The appearance of all of them warranted Oppenheim's synonym for the condition—*Dysbasia Lordotica Progressiva.*

It is apparent that changes in the static mechanism caused definite alterations in the muscle groups, especially in the hamstrings. Fixation developed at the knee and the hip. The hyperstatic condition was also seen in the inward rotation of the knee as well as in the marked plantar flexion of the foot and the toes. When studied by means of slow motion pictures the hyperkinetic overflow became evident in the soleus gastrocnemius groups. Fixation at the hip restricted the extension so that the trunk was not projected to the usual degree and in the characteristic manner. (Fig. 7.)

3. *Second Sister. Condition More Advanced. Dystonic Movements More Extensive.* The third case of the series was the most advanced of the three children in this family. The patient was a young woman twenty years of age. Her condition was the result of seven years of progressive development. The manifestations began in the lower extremities and gradually extended to the upper extremities. The case is, perhaps, unique, in that it refutes the claim of most observers that the muscles supplied by cranial nerves are not involved in the disease. The facial muscles and those of the tongue were affected to such a degree as to make the patient's speech difficult and almost unintelligible. The speech was of a drawling character, articulation being finally accomplished after efforts to overcome a pronounced hypertension, evidently caused by a hyperkinetic overflow. The extreme muscular activity seemed to be due to sustained tonic spasms in a large number of muscle groups many of which were not pertinent to the particular motor act in process. These spasms produced distortions of the body with marked deformity of motion.

At the usual cinematographic rate of reproduction the patient bore a strong resemblance in her gait, attitude and posture to the brother and sister. There was a similar flexor fixation at the knees, with an equinus fixation of the feet when the patient stood. In locomotion the right heel failed to reach the floor. The typical pronation position of the forearms was emphasized.

In slow motion pictures the analysis discloses the typical hyperkinetic and hyperstatic disturbances. The marked overflow of innervation is apparent in nearly every group of muscles in the body, producing needless and often untimely postures as well as unnecessarily active motions and movements wholly unnecessary to the acts performed. (Fig. 8.)

VI. ANALYSIS OF A CASE OF DYSTONIA MUSCULORUM DEFORMANS SHOWING EXTREME SUSTAINED SPASMS

The patient was a young man, twenty-five years of age, born in Switzerland. His family and personal histories were unimportant medically. The first indication of his malady appeared during his twelfth year when the muscles of the right leg began to act peculiarly, stiffened up and produced an awkward gait. In locomotion, as the condition advanced, the whole body would be brought forward with a "pitch or lurch" to the right.

An important feature in the history of this case was the fact that, following a long period of rest and relaxation treatment, there was pronounced improvement in the dystonia, and for several years there was almost complete cessation of abnormal movements. During this period, under conditions of intense emotion only would there be mild recurrences. From his seventeenth year, however, after an experience causing intense mental strain, there occurred a rapid generalized involvement of the somatic musculature, reaching a remarkable degree of distortion.

At the normal rate of cinematographic reproduction he appeared as a trained athlete, his muscle groups standing out prominently in their unusual development. The hypertrophy was real and the muscle strength in proportion. Testing the muscle capacity by feats of strength it was easy to determine that good muscle power was present, but the lack of proper control made it impossible for him to direct his energies to accomplish any purpose. In endeavoring to wrestle, the patient's opponent felt the full power of the groups of muscles, but the static dissociation threw the patient into involuntary attitudes which defeated his efforts and thwarted his skill. The kinetic energy appeared to develop good muscular power, but produced sustained spasms which caused perversions in all of the motor performances which he undertook. The trunk muscles were quite as much involved as those of the extremities. They threw the body forward and were augmented in their unchecked force by the upward projection of the extensors of the thigh, leg and foot. The force in extension movements was so great as literally to hurl the patient's body forward. In the previous cases attention has been called to the typical equinus tendency which forced the patient to walk upon the ball of the foot. The sudden and uncontrolled flexor and torsion spasms of the trunk with the extension of the arm, gave the impression that a heavy object was about to be hurled with great force. The incessant and powerful muscle activity with its violent contortion suggested a sudden overflow of kinetic energy causing extension of the entire body musculature. Now the flexor groups were in dominant ascending power, and again the extensors. The normally opposed groups seemed to fail in their check function in time to arrest this involuntary overflow.

The details of this extravagant expenditure of energy are revealed in bradykinetic analysis. The characteristic attitudes and poses, clearly showing how the static mechanism gives rise to the clinical pictures, are much more generalized than in any of the previous patients, but the distinguishing features of the muscular deformities are at once recognized. Indeed, if the film is stopped, almost any single picture would enable one to diagnose from its pose the nature of the malady. There is an absence of permanent contractures, conspicuous in some of the other cases, especially those of long duration. This is in part due to the almost universal involvement of the somatic groups, and probably also to the apparent absence of pathological changes in the muscles.

That the emotions exert a strong influence over these bodily movements is generally understood, and this is true of all forms of pyramidal and particularly of extrapyramidal lesions. Sitting in a perfectly quiet environment, the patient had no difficulty in inducing almost complete relaxation, and the abnormal movements ceased. He was able to feed himself and even move about slowly, with almost normal control. Being morbidly sensitive, he felt his isolation to a degree which had a decided effect upon his psyche. The slightest suggestion that he afforded amusement to others intensified his loss of control. A mental attitude developed in this case which tended to bring about an unfortunate state of conflict between his psychic state and his environment. This was not the fact in other cases. It makes necessary a further study of the psychology of the individual, especially in relation to motor control. (Figs. 9 and 10.)

VII. ANALYSIS OF A CASE OF DYSTONIA MUSCULORUM DEFORMANS SHOWING THE "DROMEDARY ATTITUDE" AND SO-CALLED SEMILUNAR FOOT

The patient was a girl of seventeen with good personal and family history. The long duration (ten years) and extreme degree of the manifestations peculiar to this disease give the case an especial interest. Born in this country of Russian Jewish parentage, the girl's first seven years of life showed normal development. At about the age of seven, peculiar, involuntary movements appeared, which were at first limited to the right foot but gradually spread to the right hand and arm. The description of these initial motor symptoms given by many of the patients suffering from this type of the disease, furnishes a conception of the character of this muscular involvement. It suggests what is understood under the term spasm.

In the course of the following three years various muscle groups became affected while involvement of the musculature of the back and neck was followed by that of both extremities upon the left side. The extension of the disease to all of the somatic groups covered a period of from three to four years, culminating in the incessant movements characteristic of her condition. The muscles supplied by the cranial nerves, with the exception of the sternocleidomastoids, and the trapezii were not involved. When in the prone position, the patient found lying on the left side most comfortable and the extreme deformities, those that were transient and those that were permanent, were then well demonstrated. The patient's movements, because of contractures and resulting deformities, were extremely slow, so that the contrast between normal and high speed photography is not so striking in this case as in others. The appearance of the patient in the cinematographic reproduction at normal speed was that of the typical *dromedary attitude and locomotion.* The extreme deformities of the feet were unusual, and had it not been for a definite history, we would have suspected a condition of congenital clubfoot.

In the slow motion pictures the characteristics of dystonia are demonstrated, though the extreme contractures and joint deformities have changed the original attitudes and now limit the play of muscular groups. Even in the normal projection and now more clearly analyzed, the movements and postures appear as overaction of certain groups of muscles, and it can be readily seen why the term dystonia has been adopted. As a matter of fact, the defect is due to hypertonia, inasmuch as the hypotonia is relative and not real. However, lordosis and dysbasia are even more obtrusive symptoms in nearly all cases. There is a fixed flexion at the hip and knee, with inversion and extension of the right foot, while the left foot is extended. The phalanges of the feet are strongly flexed increasing the marked concavity present in the plantar arches, and giving rise to the formation of what is called "semilunar" foot. So extreme are the deformities that the plantar surfaces of the feet do not touch the floor. Closer study brings out flexor-extensor movements of the fingers of the paralysis agitans type. Abnormal involuntary movements in various groups of muscles keep the body, both in the prone and the upright position, in continuous action. The muscular contractions of the various groups are so constant as to keep up a series of sustained, arrhythmical spasms. The muscles of the lower extremities were atrophied while those of the upper extremities and their axio-appendicular groups were greatly hypertrophied. The partial atrophy of the muscles of the legs was doubtless due to local changes in the joints, those of the ankles and feet being ankylosed. These were doubtless secondary mechanical results of the muscular disorganization.

Sudden tic-like movements of muscle groups in the upper extremities appeared, occasioned when the patient was under emotional strain. They appeared to be myoclonic in type. In no single case of dystonia have we seen such a combination of abnormal movements. Indeed, one might hesitate to designate this case as one of pure dystonia musculorum deformans, although it possessed the distinctive features. (Fig. 11).

VIII. ANALYSIS OF A CASE OF DYSTONIA MUSCULORUM DEFORMANS WITH PROGRESSIVE IMPROVEMENT

This patient is one of those cases of dystonia which have shown improvement by means of physical re-education. Bradykinetic analysis reveals certain deformities of motility which our previous studies have shown to be characteristic of the organic affection. The boy was normal until his eleventh year. About a year previous to this he fell from a fire-escape on the first floor of a building. He landed upon his feet and feeling no ill effects whatever, continued playing. There was nothing in the history of the following year to indicate any connection between the fall and the onset of his illness one year later. The first symptom was an outward rotation of the right lower extremity. Some two years later sudden involuntary pronation movements of the right hand, followed about a year later by an involvement of the left lower extremity, completed a picture which gave the patient the characteristic cast of the milder type of dystonia. His appearance on re-examination showed a decided improvement as compared to that of about a year previous. Nevertheless, viewed in the light of the patients already studied, there was characteristic pose and attitude of the body as a whole, and of the extremities in particular.

In the slow moving pictures the right lower extremity showed peculiar external rotation at the hip, knee and ankle, with hyperextension of the foot in sustained equinus position. Bradykinetic analysis of this motor phenomenon was, in itself, sufficient to make the diagnosis. The tendency to pronation position of the left upper extremity; the extreme extension of the toes of both feet at the same moment as the patient was about to turn, the pronounced upward shift of the entire right lower extremity, the lordotic curve with the suggestion of torsion in the trunk, were unmistakable.

The patient was next observed as he moved "on all fours." This position, as the patient moved forward, brought out even more emphatically the special deformity apparently due to defective postural fixation. Obviously, this deformity of movement would not have been apparent had he moved forward on hands and knees; the defect in attitude just alluded to would not have been brought out, since the extensors of the lower leg and foot would thus not have been brought into play. To demonstrate this fact it was necessary to observe him moving forward resting on fingers and toes and ball of foot. (Figs. 12 and 13.)

In summary, the analysis of these cases of dystonia musculorum deformans reveals the fact that an actual disorganization exists in the relation of the static and kinetic mechanisms. The marked hyperstatic defect appears to produce initial and intermediate postures of such extreme fixity that an unusual amount of kinetic energy is required to overcome them. The dystonia, therefore, seems to arise from a marked imbalance in the ratio of static energy to kinetic energy. This is particularly true during volitional effort. The pronounced hyperkinesis during inaction indicates an unstable condition in the kinetic system, more probably in that portion concerned with automatic associated movements, for the reason that the motor disorders are suggestive of movements of this kind. The disturbance in both the static and kinetic mechanisms partakes of the characteristics indicative of irritation. In addition to the evidence of irritation in these two mechanisms, the analysis points to a disturbance in the rhythm and interrelations of these mechanisms, which, being improperly synchronized in time, force and rate of action, lead to a conflict between hyperstatic and hyperkinetic innervation.

IX. ANALYSIS OF A CASE OF CEREBELLAR ATAXIA

The patient was a man forty-one years old whose defects in motility were first observed in infancy. Voluntary efforts of upper or lower extremities, or of the trunk in change of body posture, were always attended by irregular oscillating movements.

His equilibratory defects made locomotion difficult. Incoordination in the speech mechanism imparted a jerky quality to articulation which in consequence was often unintelligible.

At the cinematographic rate of reproduction, the patient demonstrated a marked ataxia in extremities and trunk. The slow motion pictures furnished opportunity for the analysis of the disturbance. When the patient attempted to walk he suddenly lost his equilibrium and fell. As the trunk approached the ground, and an effort at balance was made by the patient, the normal interplay of agonists and antagonists was seen to be defective. The segmental portions of the body as it fell did not follow in sequence nor did they retain their relationship of posture and attitude as seen in the normal. There was evident loss of associative static control. The same group dissociation was seen upon studying the efforts to rise. The extreme disproportion in muscular effort was due to lack of harmonious action between muscle groups. As he attempted to regain the upright position, he sustained for a moment his entire body weight upon his hands; taking advantage of the fact that there was no reduction in dynamic force, he attempted to hurl his trunk upward, thus to attain the upright position without going through the normal sequence of segmental attitudes. With the usual cinematographic speed the details of these pictures would have been beyond visual recognition.

A study of the details of defective motility not associated with equilibration and yet dependent upon synergic control was offered by a series of pictures showing the finger-to-nose test. The hand and finger on approaching the nose showed irregular oscillations which gradually increased toward the end of the act. The oscillations in the upper extremity were due to lack of balance between the components of the synergic units of the arm groups. In analyzing the nature of the rebound phenomenon of Gordon Holmes, in slow motion projection, the wrist when suddenly released by the examiner, rebounded forcibly striking the cheek. This rebound was due to a failure of the check component—the triceps in this case—to play its part properly in the synergic unit during flexion.

A projection study of the motor phenomenon of the pendular knee reflex shows that this is also due to an inadequate relation between the check and dominant elements in the synergic units producing flexion and extension of the leg on the thigh. Normally the impulse conveyed by tapping the extensor tendon stimulates the quadriceps extensor, the dominant element, and the posterior thigh group, which act as the check element. Working as a synergic unit the two groups during the production of the knee-jerk restrict the motor reaction to one to three oscillations of the leg. This is due to the mutual check action of the two muscle groups affected by the reflex. This fact has been demonstrated (Tilney) by kymographic records in the case of all normal reflexes when, as in this case, the synergic unit for extension and flexion of the leg is dissociated, the mutual check action is lost and this pendulum-like swinging results.

A striking feature of the bradykinetic analysis in this case was the long interval of time that lapsed between the application of the stimulus and the response as seen in the knee-jerk. As the hammer slowly descended upon the tendon of the quadriceps extensor there was a comparatively long interval of latency before the leg began to move.

In summary, the analysis of this case of cerebellar aplasia reveals the fact that the ataxia is due to a failure of the proper coordination in the synergic units of the body and that the disproportionate innervation of the dominant and check elements in each synergic unit leads to the characteristic disorder of cerebellar lesions, *asynergia*. (Figs. 14, 15, 16.)

CONCLUSIONS

1. Bradykinetic analysis by means of slow motion pictures furnishes one of the most direct approaches to a clinical study of disturbances of motion.

2. Adventitious elements in motor dis-

turbances are manifest as disorders either in the kinetic or static mechanism.

3. Cinematographic analysis indicates that all purposive movements depend upon the concurrent and synchronized operation of the kinetic and static mechanisms. Such movements are made up of a series of postures beginning with an initial posture and concluding with a terminal posture. This series of postures during a movement may readily be demonstrated by stopping the projection apparatus at each picture either in the slow or normal reproduction. Such an act as taking a step forward then appears to be composed of a series of successive postures.

4. The kinetic mechanism is represented by a neural apparatus capable of correlating the impulses necessary to the production of purposive movements, attitudes and postures, ultimately giving these their proper motor expressions. It, so to speak, prescribes and produces movements and postures for which reason it is a *kinethetic* (causing motion) system.

5. The static mechanism is represented by a neural apparatus capable of correlating the impulses necessary to the proper maintenance of postures. Ramsay Hunt has graphically illustrated the relation of posture to motion in his expression that "movements are followed by their attendant postures like shadows." Analysis by the slow moving pictures verifies this conception. The static mechanism, so to speak, controls the postures which follow movements like shadows, stabilizing them through all their phases. In this sense and because it maintains posture, it is a *statothetic system.*

6. From such evidence as is at present available, it seems warranted to conceive of a static arc representing the statothetic system with its chief center in the cerebellum, and a kinetic arc representing the kinesithetic system whose centers are in the endbrain. The trend of recent opinion would allocate the primordial center for the kinetic arc (paleokinesis—Hunt) to the corpus striatum or basal forebrain ganglion.

7.The disorders of motion investigated in this study, including choreiform movements in chronic degenerative chorea and astasia abasia, cerebellar ataxia in a case of cerebellar aplasia, intention tremor in multiple sclerosis, and dystonia in dystonia musculorum deformans, may be attributed to disorganizations in the two fundamental neural arcs already described.

8. Choreiform movements in degenerative chorea, upon analysis, appear to be due to a hyperkinetic disorder resulting in the sudden productions of postures and attitudes directly expressive of the emotions of fear, anger, disgust, etc. The static mechanism is affected to a lesser degree as shown in the instability of posture indicating an imperfect association of the synergic units. Choreiform movements in astasia abasia are likewise based principally upon hyperkinetic defects. The resulting disorders of motion produce postures and attitudes expressive of the emotions. In this case these emotions are more highly complex than in degenerative chorea, and indicate a more acutely conscious and psychic elaboration of fear.

9. Cerebellar ataxia and intention tremor, upon analysis, both appear to be due to essentially the same defect limited to the static mechanism. The motor disturbances in these conditions are produced as a result of a disorganization in the proper relation of the synergic units of the body.

10. The dystonias, upon analysis, are clearly revealed as hyperkinetic and hyperstatic in nature. The normal concurrence in these two streams of static and kinetic innervation is imperfect or wanting. These mechanisms appear to be affected by pronounced irritation or lack of proper inhibition.

11. The foregoing suggestions on the basis of bradykinetic analysis are offered with the desire of furnishing an anatomical and physiological background for certain motor disorders and at the same time of calling attention to the seemingly essential relationship of the static and kinetic mechanisms in all purposive motion. Much further study is needed in the development of these conceptions before they may be advanced as more than working hypotheses.

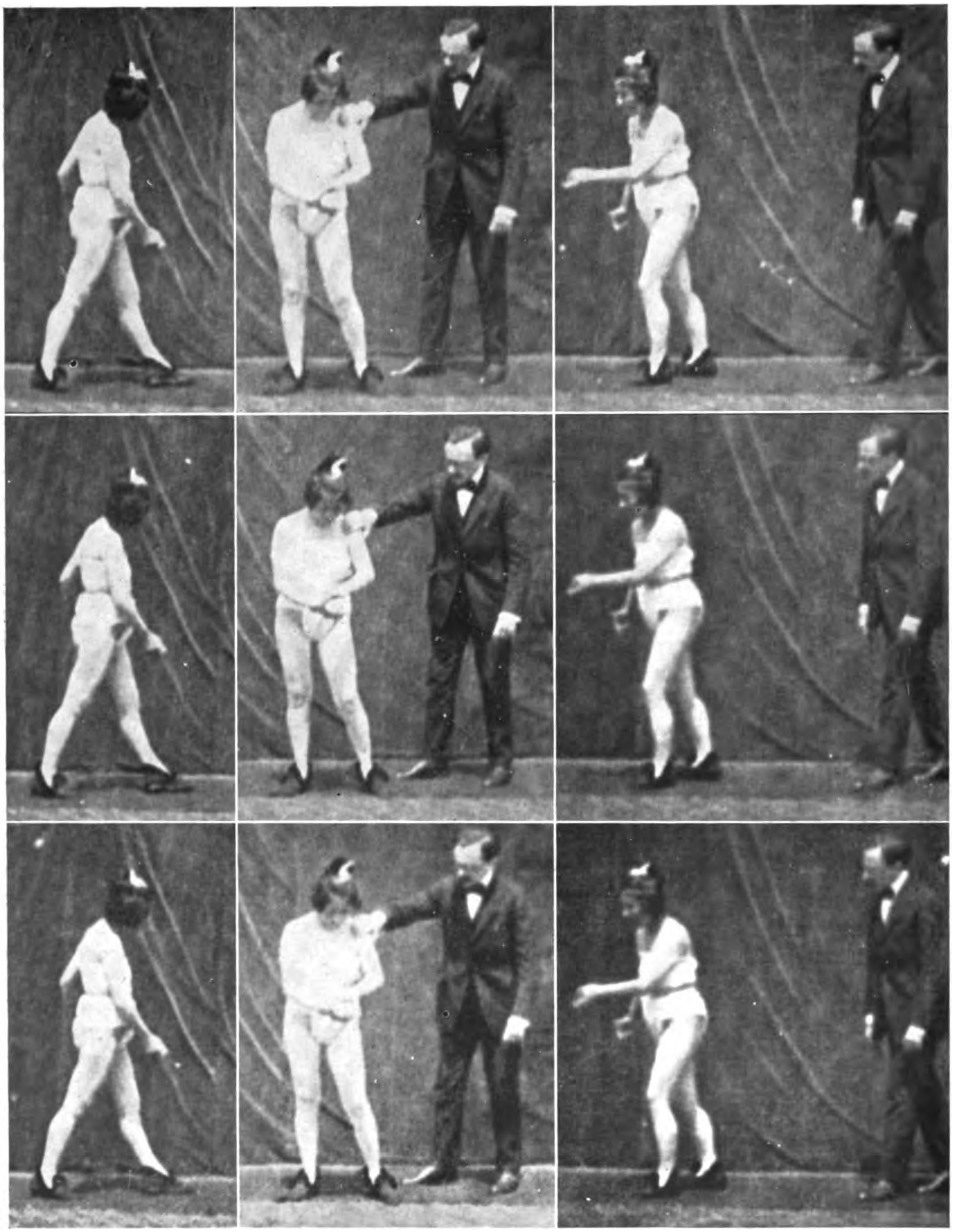

FIG. 1. Bradykinetic Analysis in a Case of Chronic Degenerative Chorea with Athetotic, Choreiform and Attitudinizing Elements.

What appeared by normal projection as purposeless, meaningless movements, by bradykinetic analysis are seen to be gesticulations, expressions of definite acts of going, of giving; and attitudes and gestures symbolizing emotions of shame, coyness, turning away, etc. The hyperextension of the left leg at the knee is seen in the picture. Athetotic (polypoid) movements of the fingers are indicated. Facial grimacing is indistinctly discernible in the photographs though clearly seen on the screen.

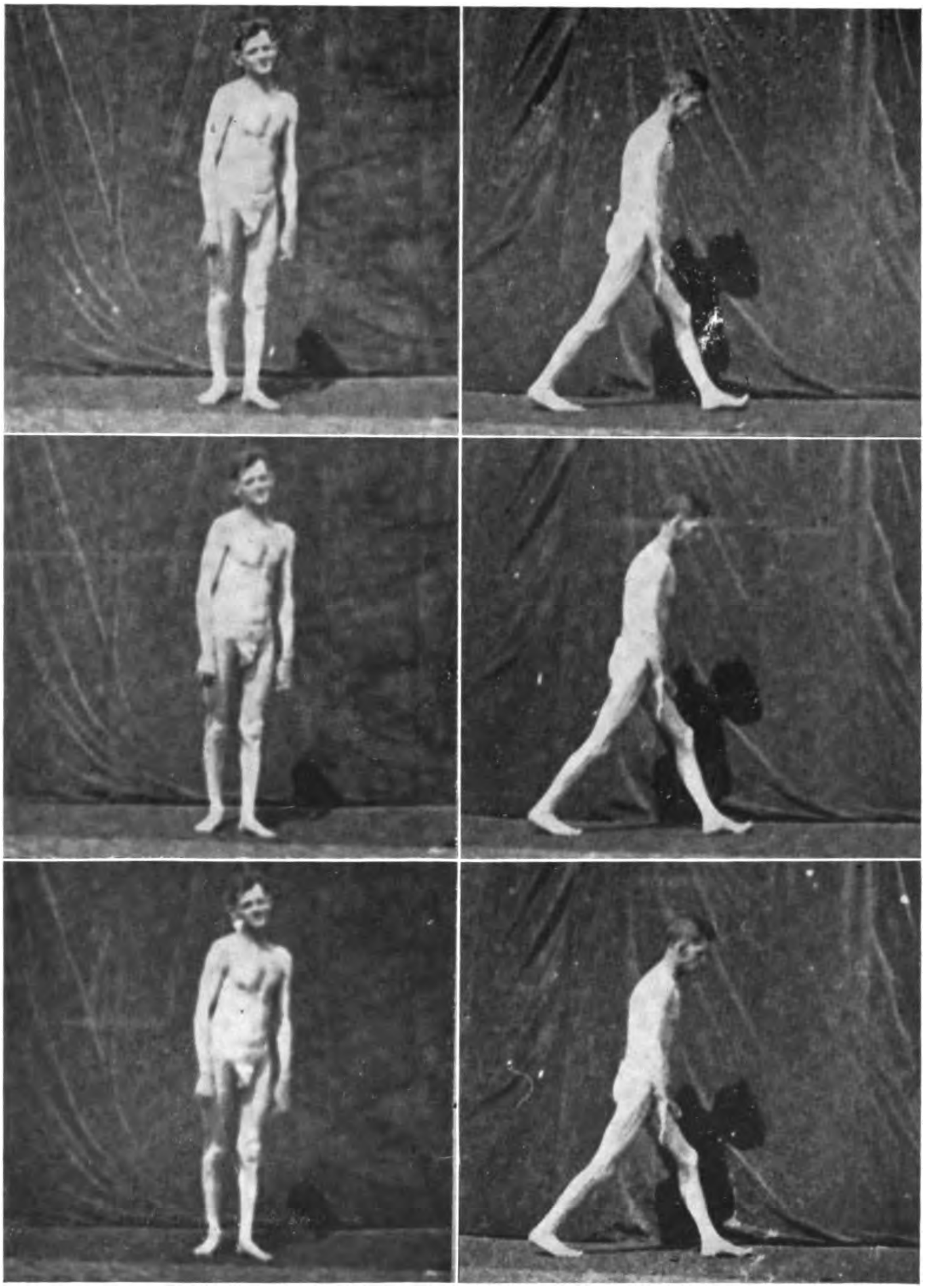

FIG. 2. Bradykinetic Analysis in a Case of Chronic Degenerative Chorea with Some Elements Suggestive of Dystonia.

Torsion movement involving upper truncal and thoracic musculature: pronator position of hand and forearms. Extensor position of big toe—not a Babinski, however. Overextension of foot in walking, almost a constant feature of gait in dystonia, is not seen in above: on the screen, however, it is occasionally observed in this patient but to a far less degree and inconstant. Some grimacing and continual choreiform movements are of a type present in chronic degenerative chorea. The torsion of the trunk involves rather the upper truncal segment in contradistinction to that seen in dystonia in which there is pelvo-truncal torsion.

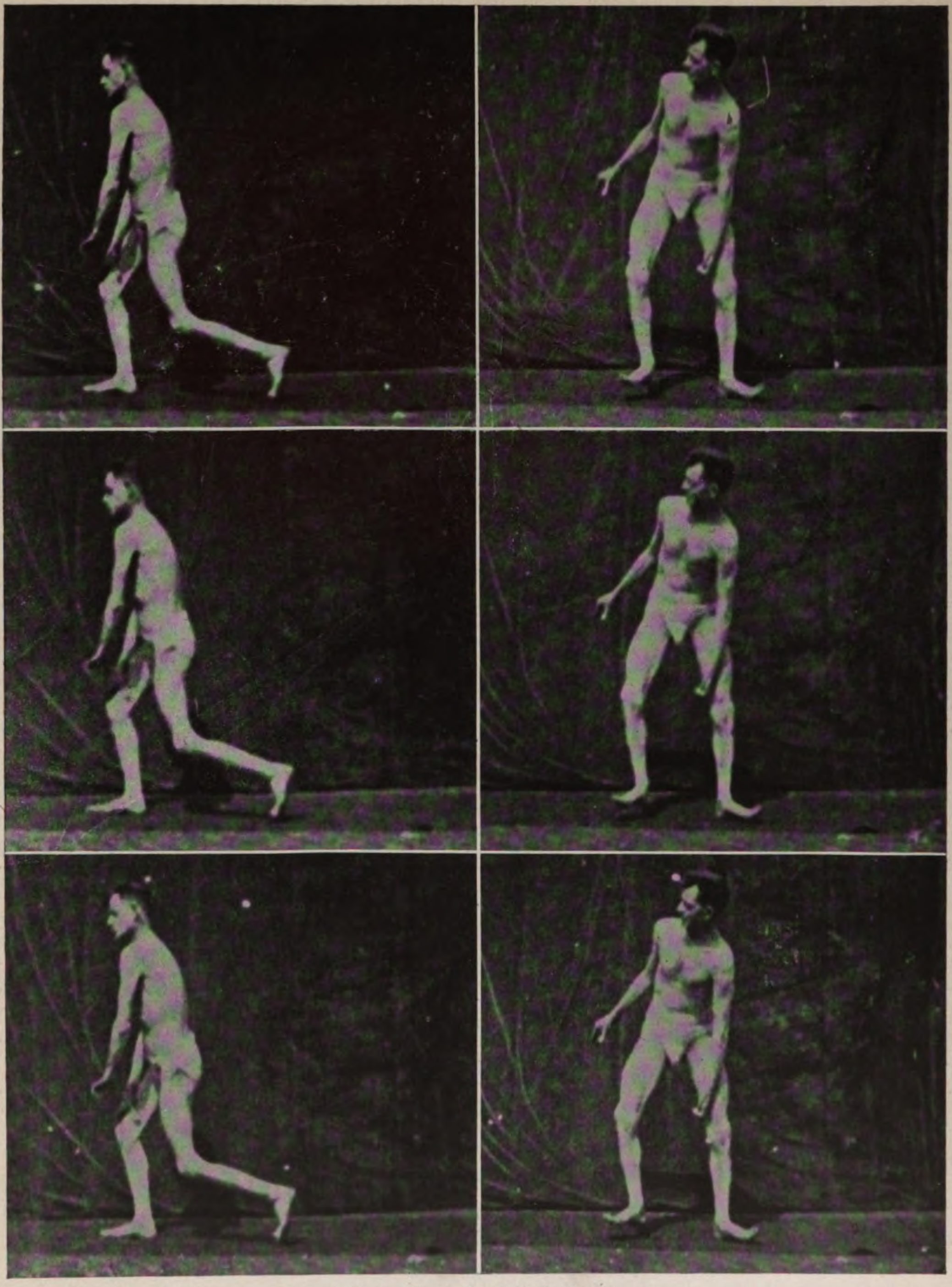

FIG. 3. Same Case as figure 2.

Gait with overflexion at the knees and pronator position of forearm with right arm fixed on thigh during locomotion give dystonic character to attitude; attitudinizing as seen in act of hurling an object. (Compart with figure 9).

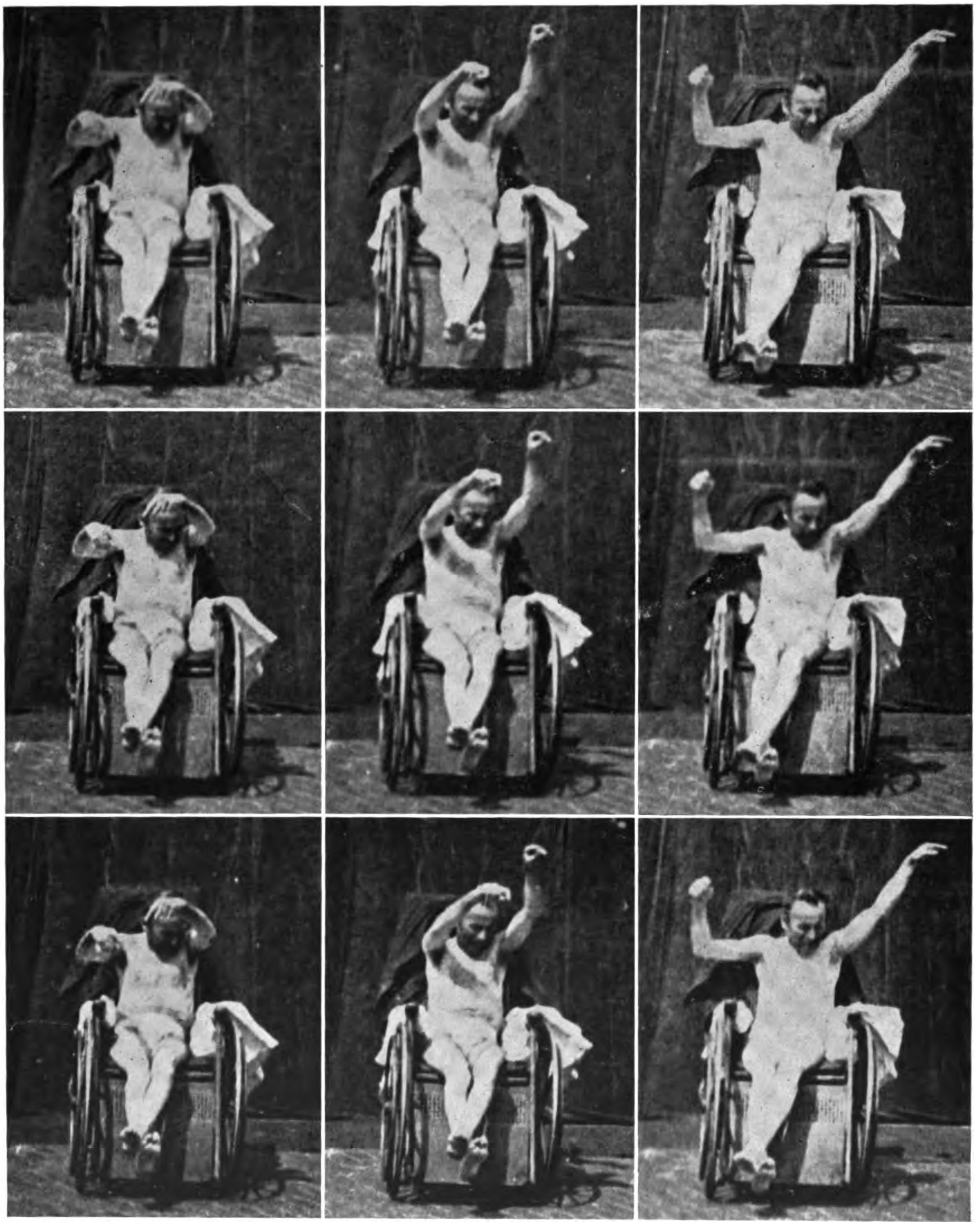

FIG. 4. Bradykinetic Analysis in a Case of Multiple Sclerosis of Dyssynergic Character. Finger-to-Finger Test; Dissociation of Synergic Unit.

Observe dyssynergy even as patient attempts to place his upper extremities in position for approximation of the widely separated digits. The defective morphology of group movement becomes more evident as patient attempts to bring his fingers together. The arms shoot far beyond the point desired, each element of advance showing the character of the deformity of motion, the dissociation of the synergic units. The lower extremities and trunkal muscle groups are all involved, the case being one of so-called integrative asynergia.

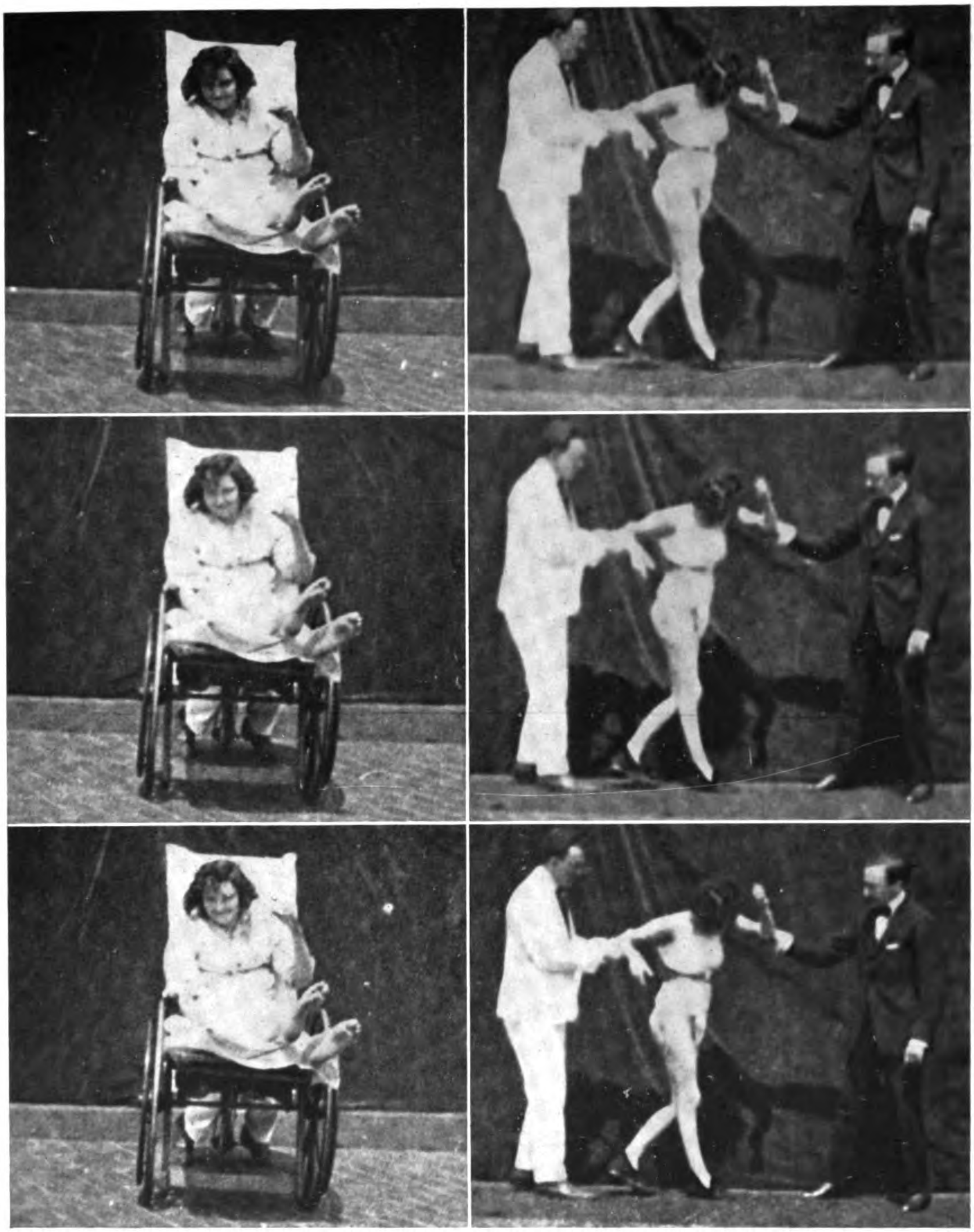

FIG. 5. Bradykinetic Analysis in a Case of Astasia-abasia with Excessive Motor Agitation.

Motor manifestations classically those of psychic origin. With patient seated the foot and toes show tendency to plantar contraction there being no paralysis of extensors and no true contractures of flexors. The upper and lower extremities are in constant movement showing distinctly a pattern; static and kinetic mechanisms act normally and in unison. Upper extremities are engaged in rythmic movements imitative of those of flying. Vermicular wave described in text is not discernible in photograph.

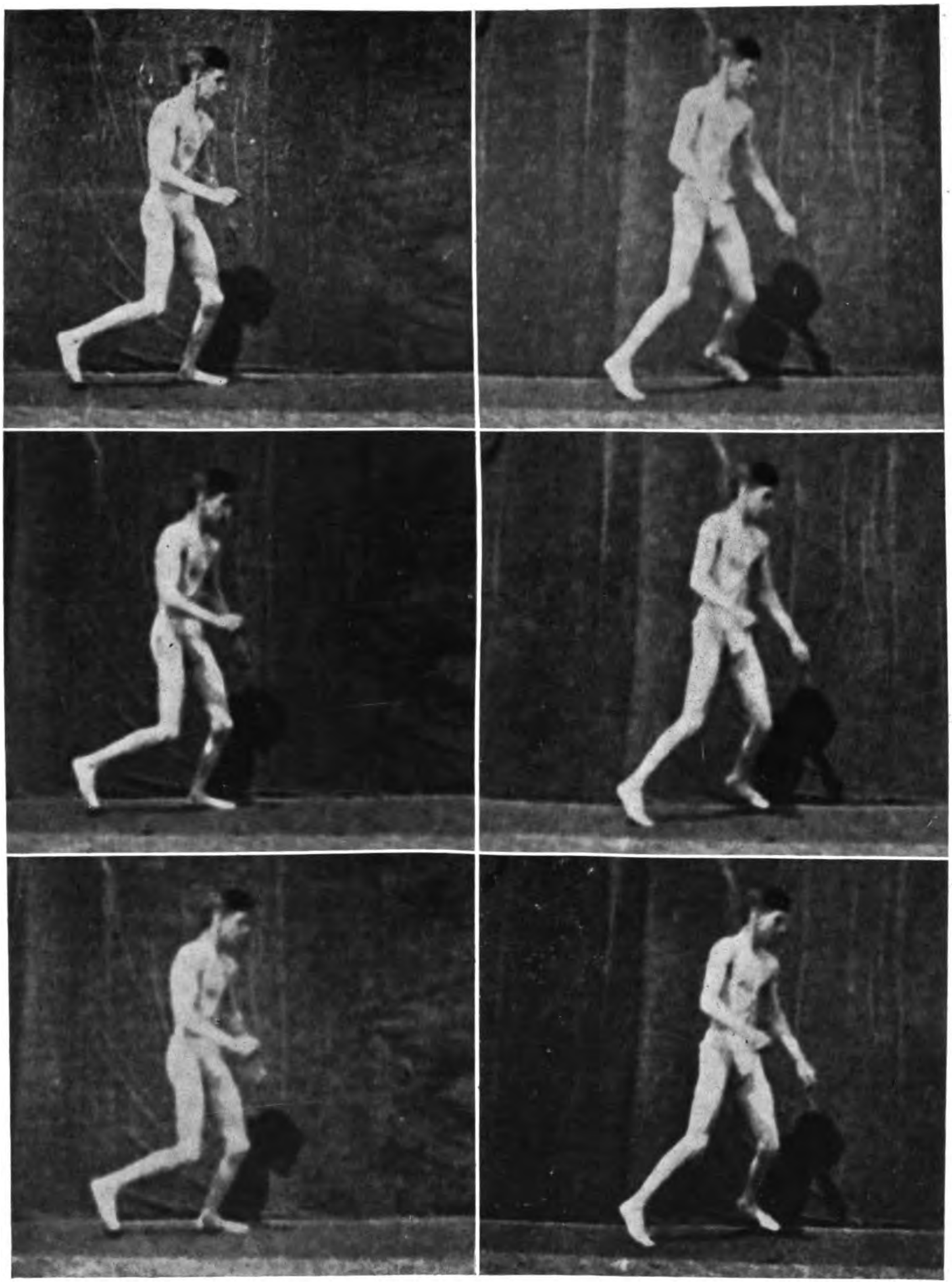

FIG. 6. Bradykinetic Analysis in a Case of Familial Dystonia Musculorum; Group of Three Members of One Family: A Brother and Two Sisters.

The above picture is that of the brother, the last to be afflicted, and showing the incipient stage. Characteristic inward rotation at knee, equinus position, overextension of foot in action with accompanying flexion at hip and knee. Extreme pronation position of forearms and hands not due to contractures.

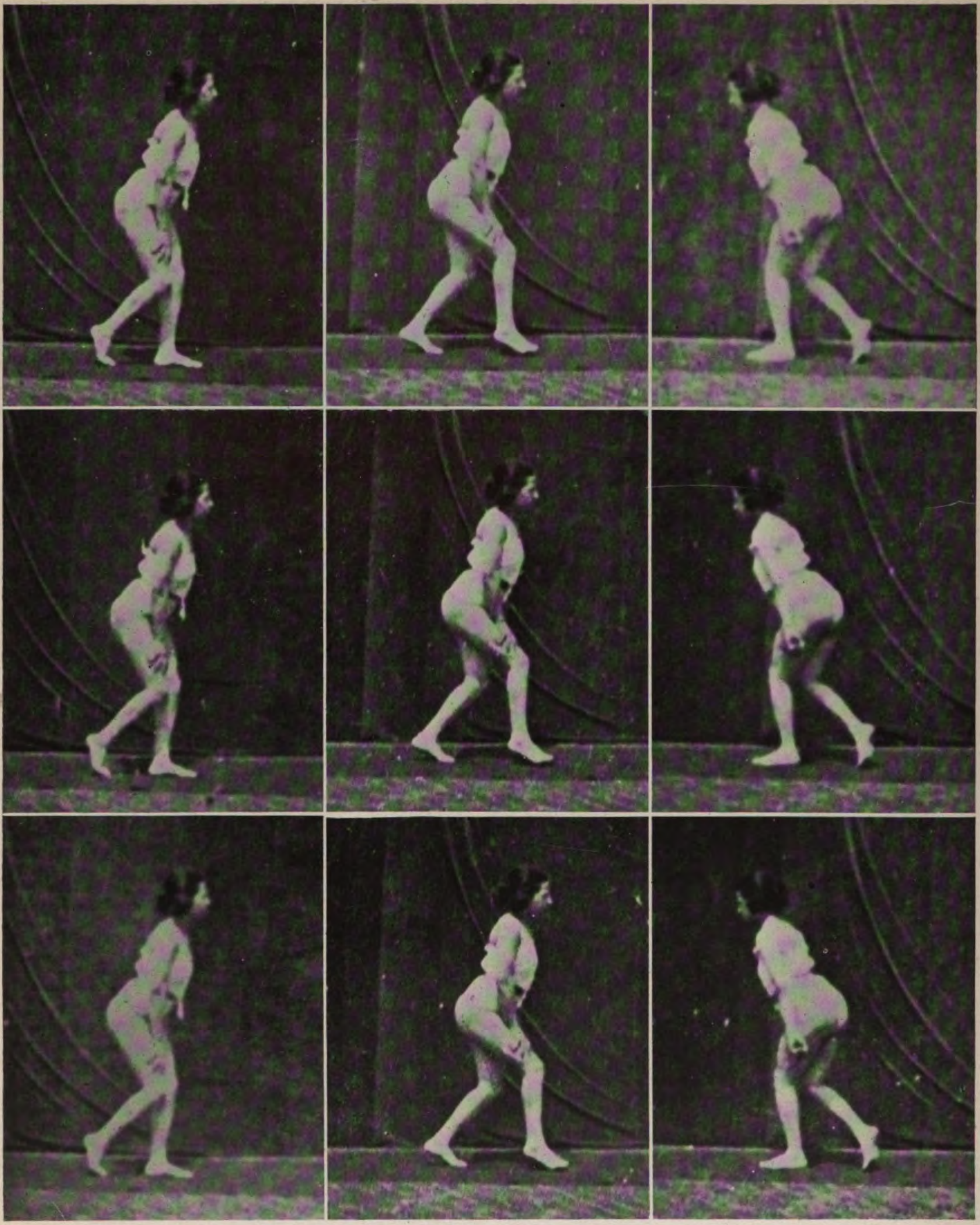

FIG. 7. Bradykinetic Analysis in a Case of Familial Dystonia Musculorum; One Sister.

Symptoms more pronounced than in the case of the brother (preceding picture). Observe lordosis, flexion at hip and knee. Flexure contractures of right hip and knee joints are fixed by muscle changes. Inward rotation knee and hip, also hyperextension at ankle while walking, together with pronator position of forearm and hand are shown in the picture. The mechanism in muscular group action is brought out in the animated series on the screen.

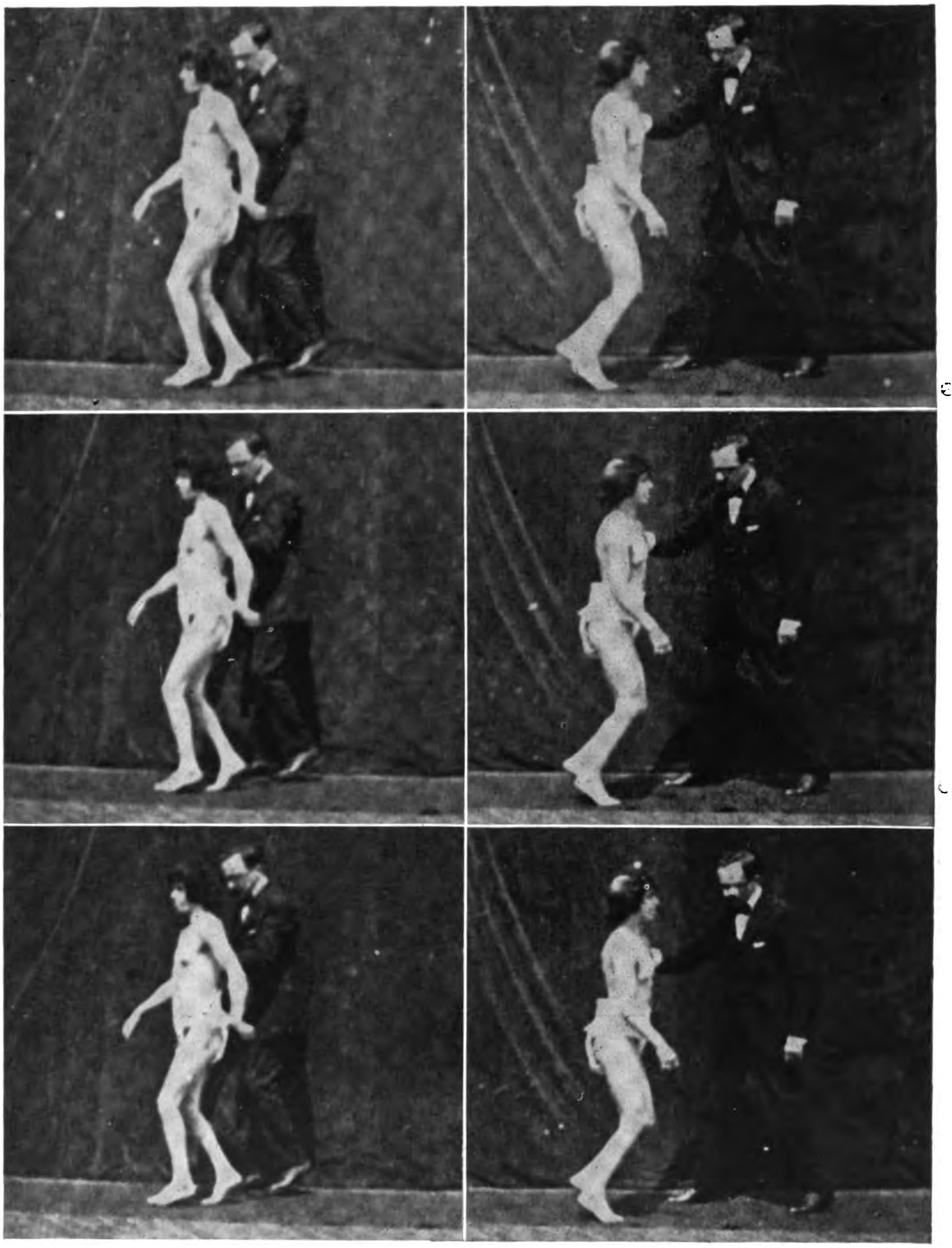

FIG. 8. Bradykinetic Analysis in a Case of Familial Dystonia Musculorum: The Other Sister.
Flexor fixation at knee; equinus position of feet; typic al pronator position forearms; inward rotation knees.

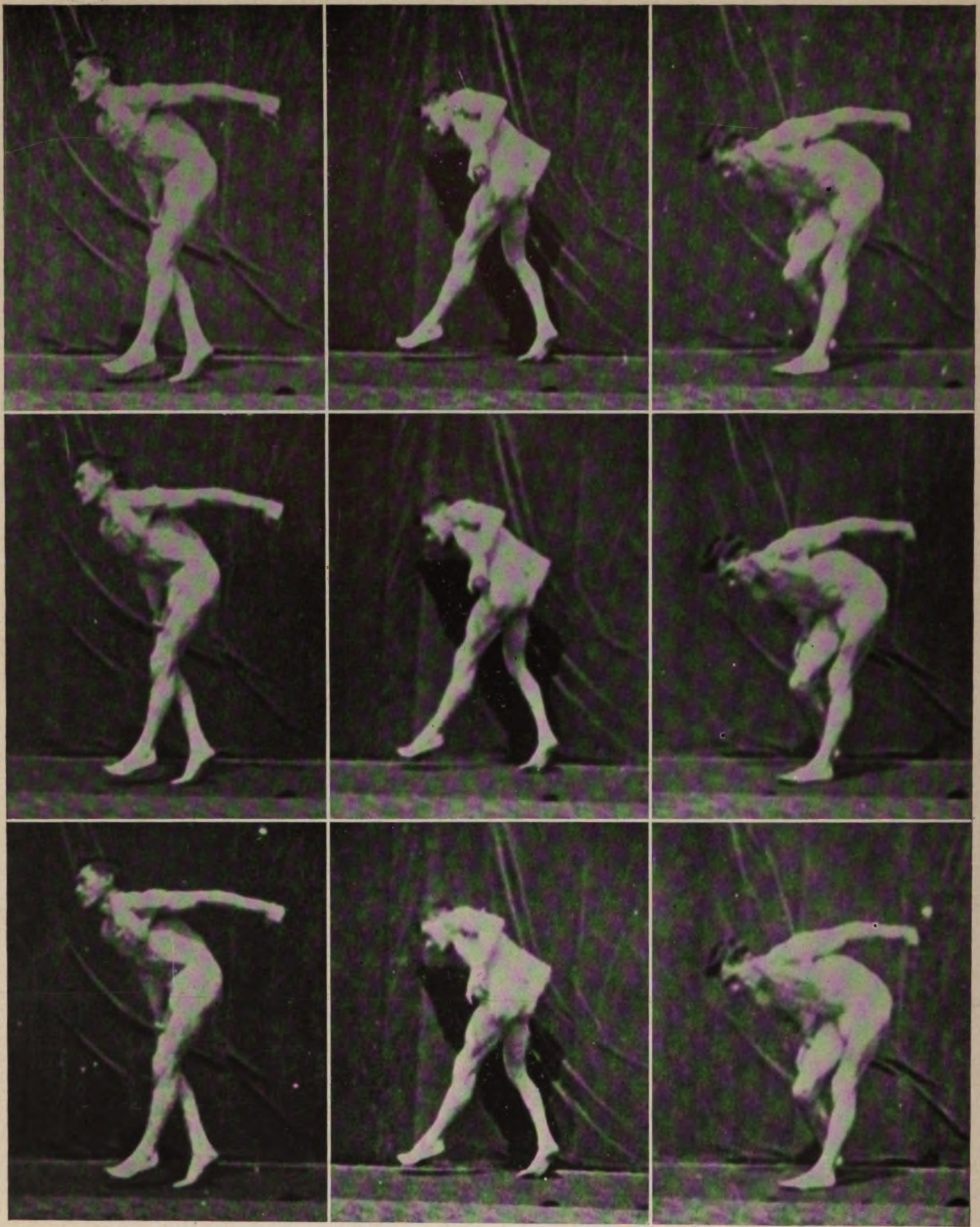

FIG. 9. Bradykinetic Analysis in a Case of Dystonia Musculorum Deformans. Involvement of All Groups of Muscles and True Hypertrophies.

Bizarre distortions of movement. Characteristic positions. Plantar flexion at ankles in walking. In some position inward rotation at knee. Trunk muscles also involved. Body thrown violently forward and movement intensified by upward projection by extensors of hip, knee and ankle. Hand resting on thigh to restrict extreme sudden flexion at hip. Some tendency to pelvic torsion. Force in extension movements so great as to literally hurl the body forward. Sudden uncontrolled flexor and torsion movements give impression patient is about to hurl heavy object. Suggestion of sudden release of kinetic energy not controlled in static fixation. There is no permanent fixation by contractures in this case.

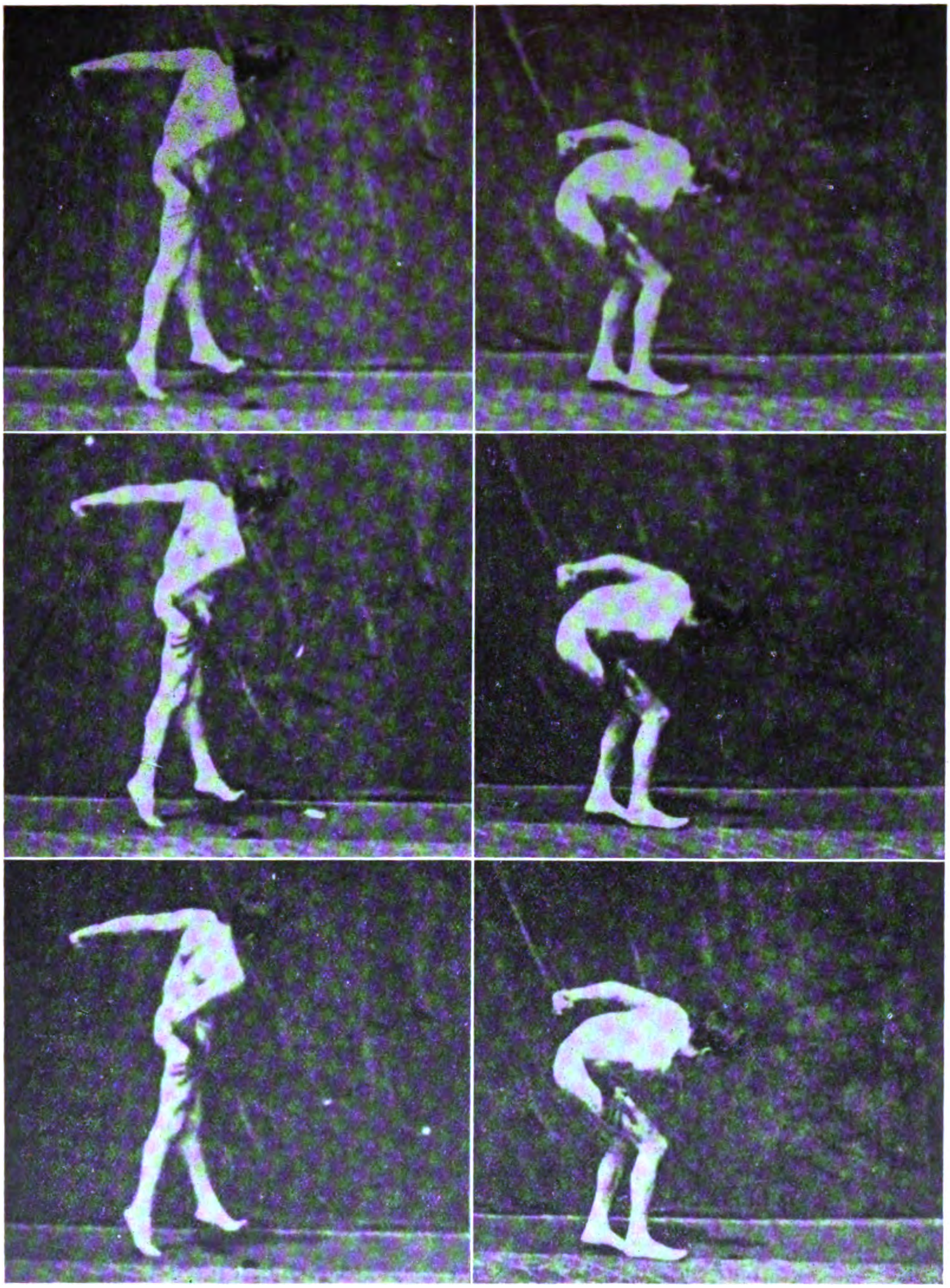

FIG 10. Bradykinetic Analysis in a Case of Dystonia Musculorum Deformans.

Same as figure 9. Observe her occasional hyperextension of toes,not often seen in dystonia.

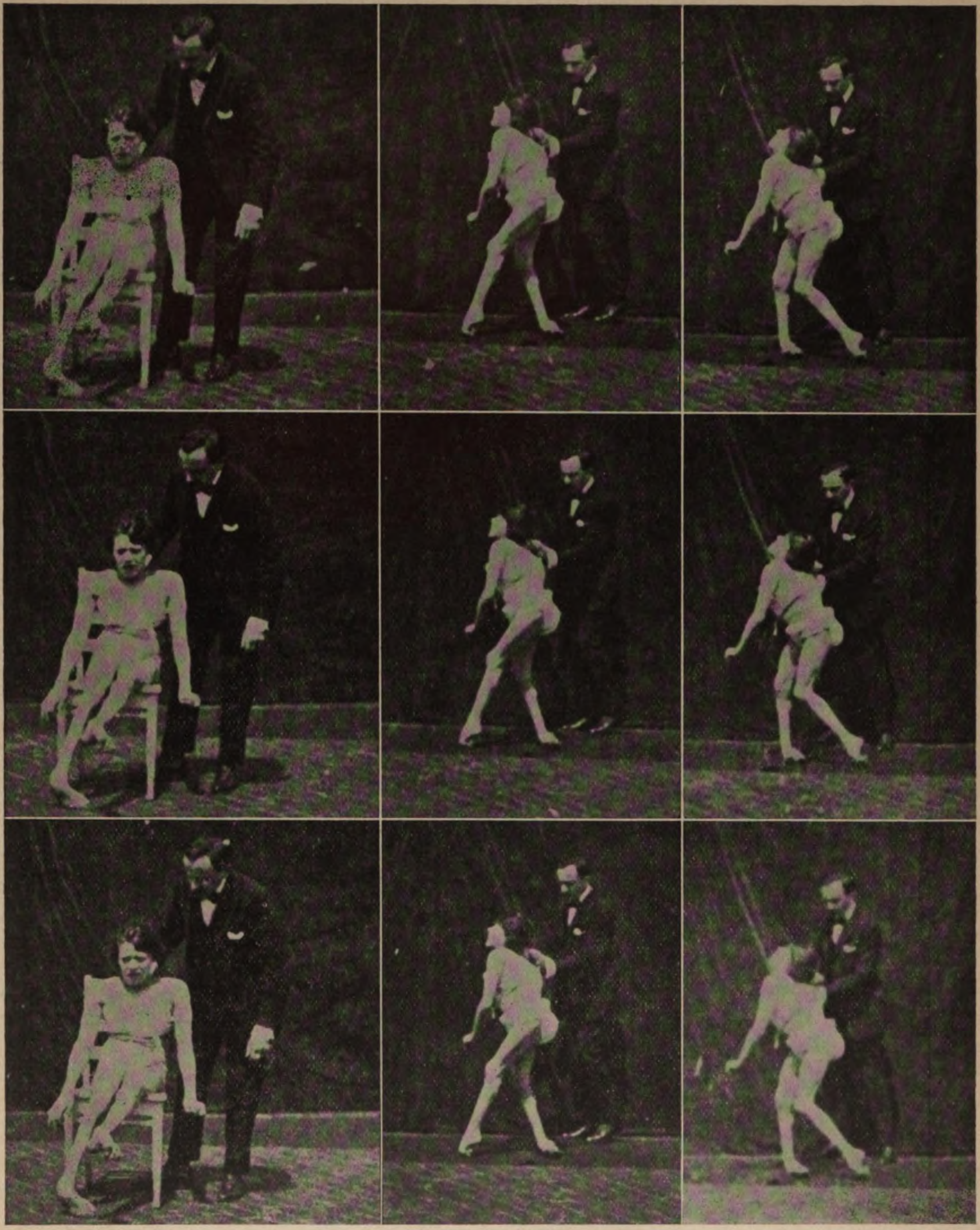

FIG. 11. Bradykinetic Analysis in a Case of Dystonia Musculorum Deformans with "Dromedary Attitude": Semi-lunar Foot.

Extreme deformities of feet; girl walks on dorsal surfaces of feet. Extensor and equinus positions have become "fixed," in less intense degree "fixation" positions in flexion have also developed at hip and knee. Lower extremities below knee relatively atrophic from partial disuse after ankylotic changes though the tibial and calf groups are still in constant myotonic activity as far as the joint changes permit. Upper extremities are in constant motion and all muscles hypertrophied. The trapezii are especially active causing ceaseless clonic extensor movements of the head. There is extreme deformity of trunk due to fixation by domination of certain muscle groups of the trunk and pelvis. Forearms are in characteristic positions. In the moving pictures a distinct rhythm of the paralysis agitans type can be observed in the movements of the forearms and hands.

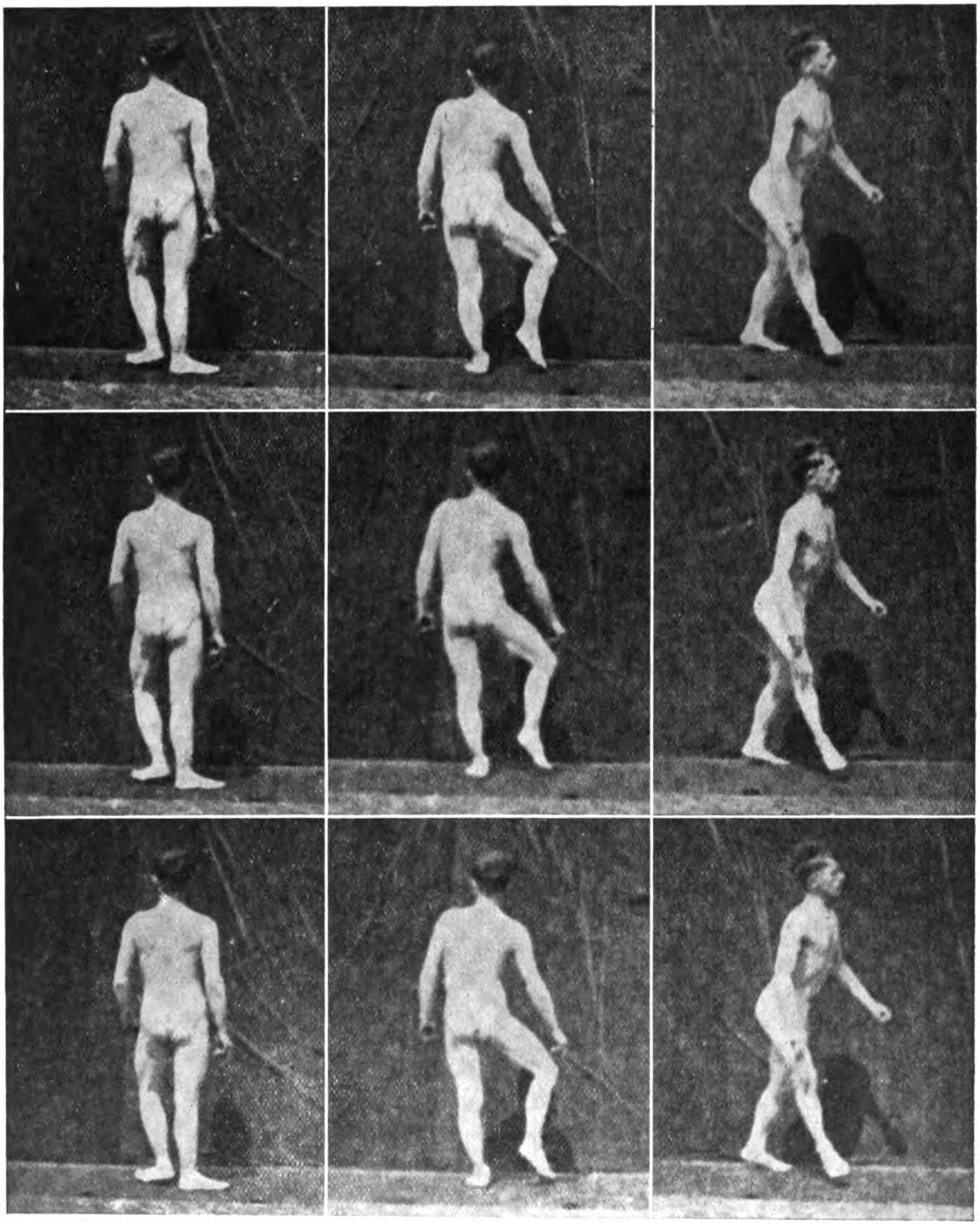

FIG. 12 Bradykinetic Analysis in a Case of Dystonia Musculorum Deformans; Marked Improvement.

Characteristic attitudes: external rotation of hip and knee, plantar flexion of foot, occasional eversion of foot.

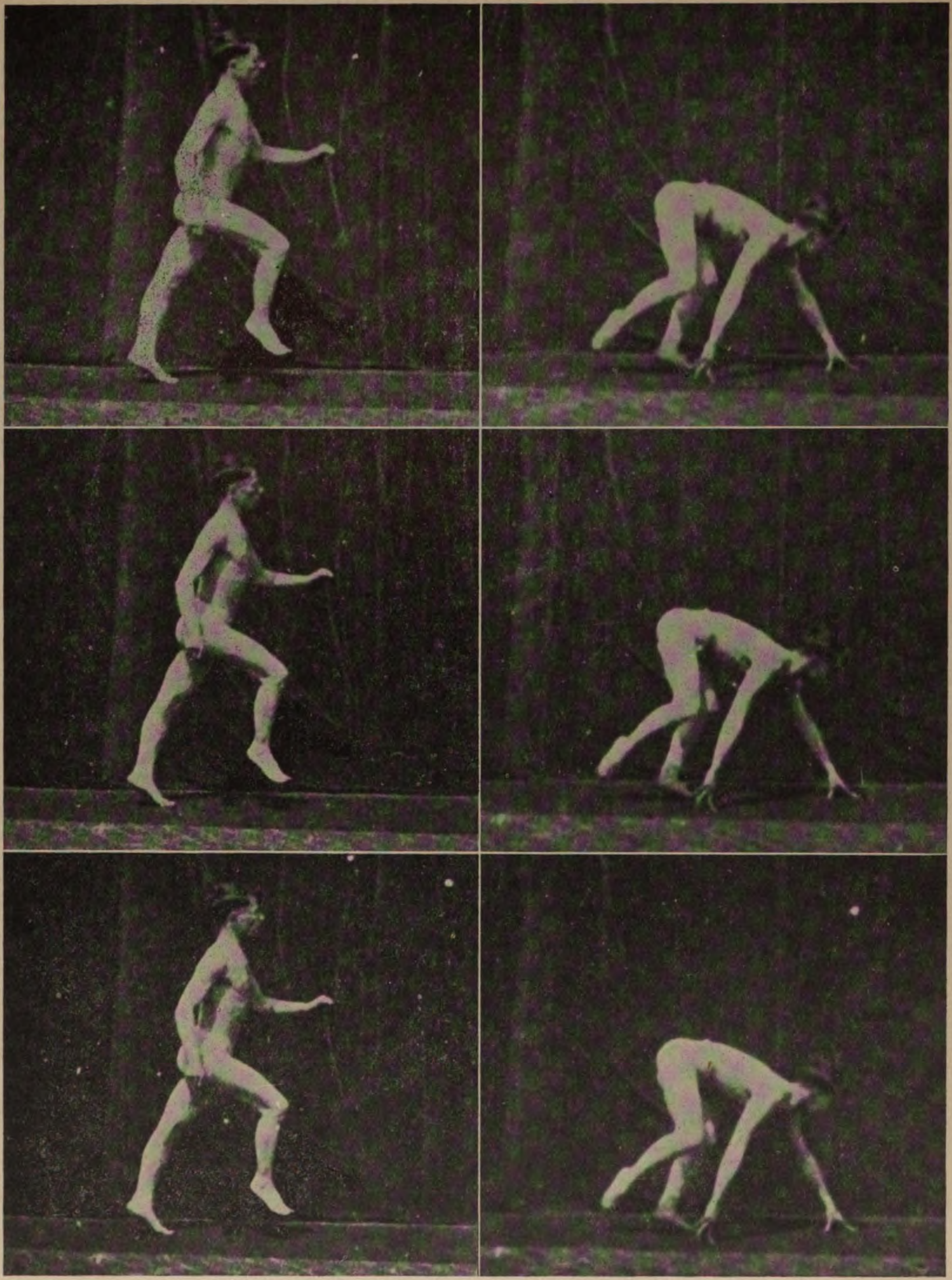

FIG. 13. Same as figure 12.

Showing marked improvement of muscle control and type of exercise helpful in re-education; demonstrating also as patient advances resting on fingers, ball of foot and toes, characteristic dystonia attitudes. This position on "all fours" as patient moves forward emphasizes the special deformity due evidently to defective postural fixation in the motor conflict which shoots, so to say, the pelvis and trunk upward in progression.

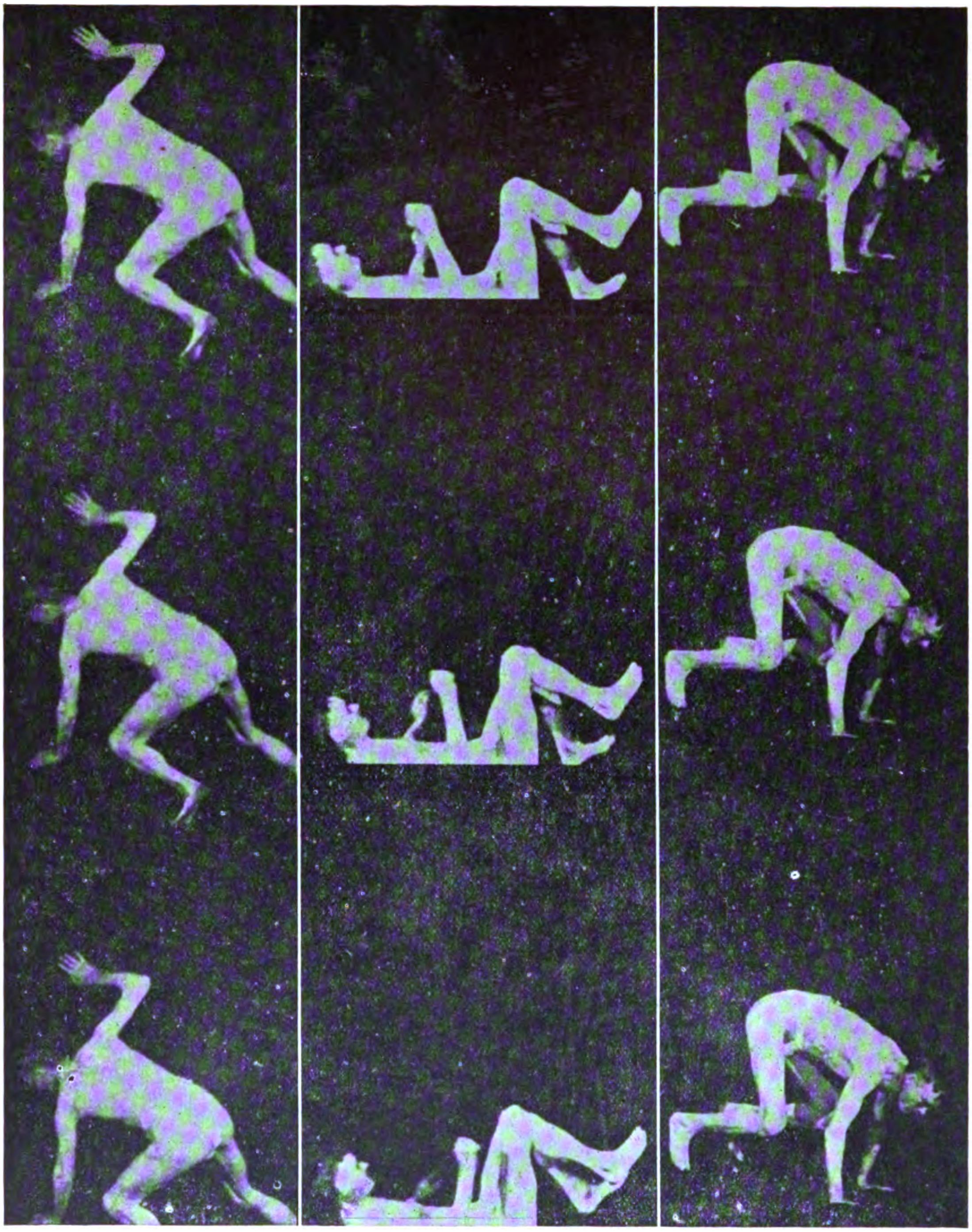

Fig. 14. Bradykinetic Analysis in a Case of Cerebellar Agenesis: Patient Falling and Rising.

As trunk approaches ground and efforts to balance are made, the normal interplay of muscle groups, agonists and antagonists, is seen to fail. Some group dissociation is seen upon efforts to rise. As patient attempts to gain upright position the defective integrative synergia causes vicarious efforts; the body is tossed upward and sustained for a moment upon the hands alone.

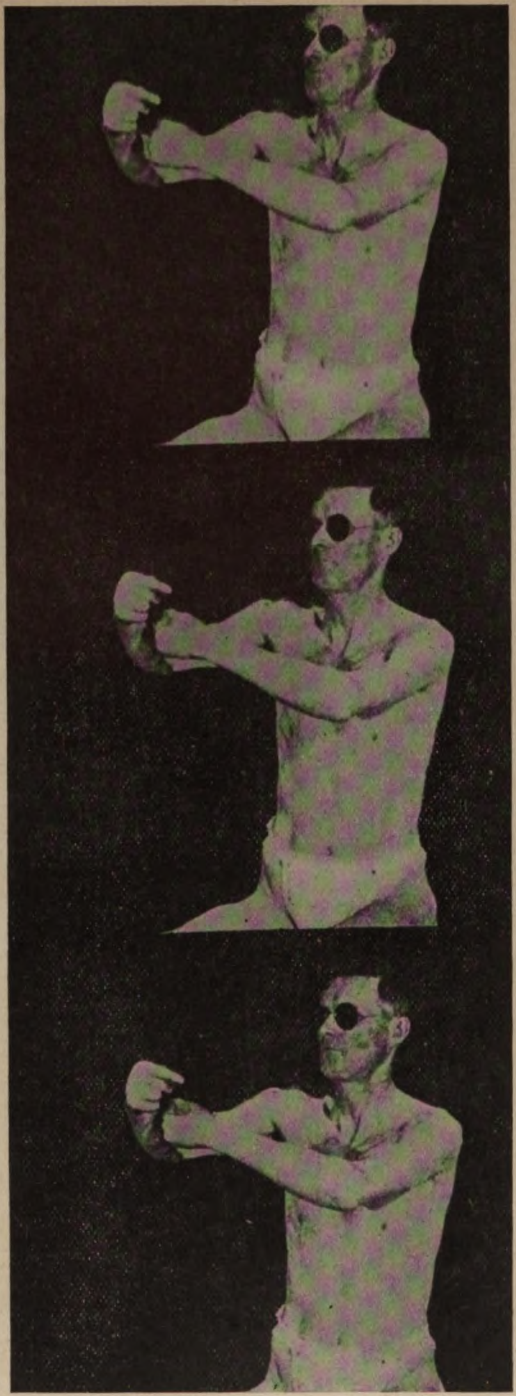

FIG. 15. Same Case as Figure 14; Showing Finger to Finger Test.

Careful observation shows the so-called tremor of the multiple sclerosis type to be the result of defective cerebellar function. The analysis by slow motion photography clearly defines the dissociation of the synergic unit. The serial pictures, of which the above illustrations are necessarily only fractional units, show the defective interplay of the agonist and antagonist and its corresponding check component thus mechanically defeating attainment of the purposeful movement.

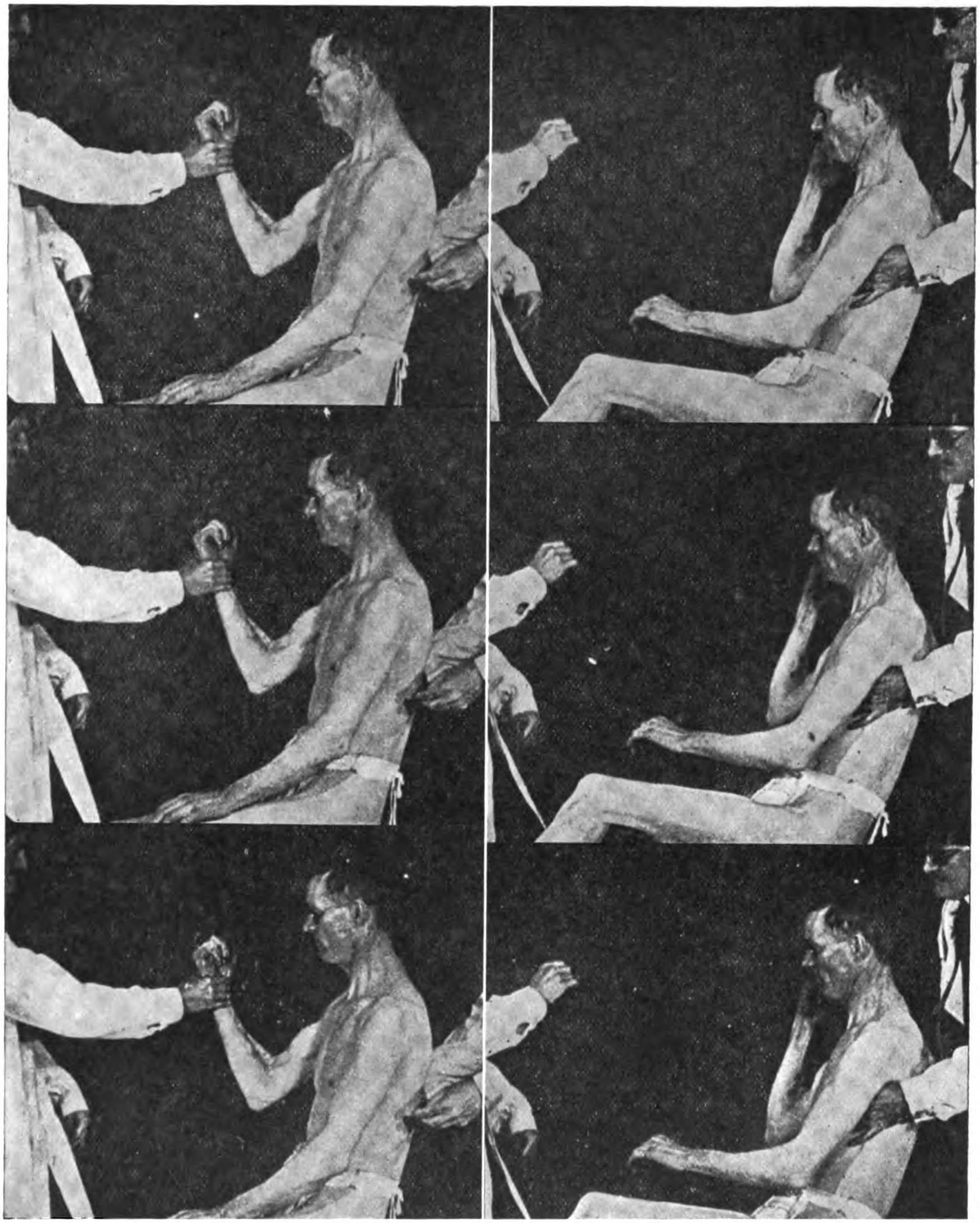

FIG. 16. Same Case as Figure 14: Showing the Abnormal Rebound Phenomenon of Gordon Holmes.

As the examiner grasps the patient's wrist and attempts to extend the flexed arm of the latter, the patient at the same time resisting, as the examiner suddenly releases the wrist, the patient's forearm rebounds forcibly, striking his cheek. This rebound, by analysis in slow motion, is seen to be due to failure of check component (the triceps) to promptly enter into coordinate function. Furthermore as indicating a general dissociation of synergic muscle group function, observe that all the segments of the body fail in associative activity resulting in general disequilibrium. (integrative dyssynergia).

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